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Case Report

Polydipsia-induced hyponatremia and status epilepticus in a schizophrenia patient: A case report from the emergency department

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ABSTRACT

Related to severe hyponatremia, various symptoms, such as confusion, vomiting, changes in mental state, status epilepticus, and a variety of conscious disturbances are sometimes seen in the emergency department (ED) but seldom recognized as water intoxication in the early stages. Status epilepticus is an emergent and life-threatening condition if not diagnosed and managed promptly and efficiently. The authors reported a case of a 31-year-old woman with history of schizophrenia with poor drug compliance. The compulsive drinking of more than 15,000 mL of water per day for 4 days resulted in intermittent episodes of vomiting and seizures and she was sent to our ED by ambulance. Water intoxication with severe hyponatremia ($[Na^+] = 112$ mEq/L) was diagnosed and hypertonic sodium supplements and airway protection were then offered. Detailed history taking and early detection of hyponatremia is crucial to prevent fatal complications of water intoxication. Emergency physicians should be suspicious of hyponatremia from water intoxication in psychiatric patients with neurologic symptoms during the observation period in the ED.

1. Introduction

Associated with a mortality rate of as high as 50%, severe hyponatremia is common in acutely ill patients and causes primarily cerebral edema and central nervous system dysfunction [1,2]. Water intoxication is an unusual but potentially lethal cause of hyponatremia. The greatest concern of all involving severe hyponatremia is cerebral edema, which can manifest as nausea, headache, confusion, lethargy, convulsions, seizures, coma, or death. Related to hyponatremia, status epilepticus is sometimes seen in the emergency department (ED) [3–5]. It is an emergent and life-threatening condition if not diagnosed and managed promptly and efficiently. Substantial amounts of patients with psychiatric disorders reveal polydipsia and polyuria without identifiable underlying medical causes, and multiple factors have been implicated, including a hypothalamic defect and adverse medication effects [6]. We report a schizophrenic and polydipsic patient with severe hyponatremia resulting from excessive water consumption and recurrent episodes of seizures in an emergency setting.

2. Case report

A 31-year-old female with a history of schizophrenia for 6 years, who has been poor compliant to regular treatment ever since her confirmed diagnosis, quit medication by herself about 2 weeks ago and was then sent to our ED by ambulance because of seizures and loss of consciousness. Her actual medical history was unknown and we could only obtain fragments of information from her family. Detailed interviews with her family revealed that she had drunk water compulsively in vast quantities, estimated at more than 10 bottles of commercial bottled drinking water (1,500 mL per bottle) per day for 4 days, and this was interspersed with episodes of vomiting, conscious disturbances, and seizure. Her vital signs on arrival showed body temperature of 36.7°C, pulse rate of 82 beats per minute, respiration rate of 21 breaths per minute, and blood pressure of 132/68 mmHg. Physical examination revealed a condition of coma with a Glasgow coma scale of E1V1M3, normal pupillary and Babinski's reflexes, no jugular vein engorgement,

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supple neck, clear breathing sound, soft abdomen, and no pitting edema of the legs.

One dose of lorazepam (0.044 mg/kg) and phenytoin (20 mg/kg) were given to control her seizure but seizures persisted to occur after initial treatment, so four additional doses of lorazepam were further offered in 1 hour. She was then intubated and supported by mechanical ventilation. Brain CT was later arranged to exclude intracranial lesions and this revealed mild brain edema. The hemogram and biochemical data of blood, including serum blood urine nitrogen, creatinine, sodium, potassium, and osmolarity were checked and reported as normal except for an obvious case of hyponatremia ($[Na^+] = 112$ mEq/L, normal range is between 133 and 153 mEq/L [hospital laboratory range]). The following laboratory data were also collected and showed a urine osmolality of 85 mOsm/kg (normal range: 50–1,300 mOsm/kg), urine sodium of 0.83 g/d (normal range: 4–6 g/d). According to the clinical presentation, laboratory data, and detailed history, hypotonic hyponatremia (water intoxication) was diagnosed, which indirectly induced the onset of status epilepticus. With an initial infusion rate of 30 mL/hr under careful observation, 3% saline solution was immediately administrated for the correction of the hyponatremia. Serum sodium was checked every 2 hours to monitor and to avoid a sudden rise in serum sodium [7,8]. No more seizure attacks occurred during the administration of 3% saline solution.

Twenty-four hours later, her serum sodium level rose back to 133 mEq/L, and her consciousness then improved for a short period of time was after which she was extubated. After recovering from disturbed consciousness, she was subsequently discharged 5 days later without any sequelae and scheduled for psychiatry outpatient department. When followed up at the psychiatry outpatient department 2 weeks later, her serum sodium and serum osmolarity were all at normal levels.

3. Discussion

This case was diagnosed as status epilepticus and was improved with the administration of sodium. After admission, a drug-induced or drug-related polydipsia was suspected. Clinical conditions that result in water intoxication include psychogenic polydipsia (self-induced), pregnancy, alcoholism, tumors (altered physiologic states), and transurethral resection of the prostate syndrome (iatrogenic) [9]. The most common causality of all is psychogenic polydipsia (compulsive excessive water consumption), which is sometimes associated with either mental illness or being mentally handicapped [4,5].

Severe emotional (e.g. psychosis) or physical stress (e.g. surgery) and various pharmacological agents are reported to stimulate antidiuretic hormone release and to be associated with the syndrome of inappropriate antidiuretic hormone secretion, which results in hyponatremia [1]. Among those medications, barbiturates, antipsychotics, and antidepressants are frequently associated with syndrome of inappropriate antidiuretic hormone secretion and most possibly offered to a psychiatric patient. In this case, because of an unknown medical history and treatment to her mental illness (e.g. schizophrenia), the hyponatremia and status epilepticus as its sequela were attributed to water intoxication.

With all the clinical findings and the laboratory data ($[Na] = 112$ mEq/L, urine osmolality of 85 mOsm/kg, and urine sodium of 0.83 g/d), a diagnosis of hypotonic hyponatremia was initially made. The patient developed symptomatic hyponatremia after consuming an extraordinarily large amount of water over a relatively short period of time (4 days). Normally, the kidneys are able to excrete in excess of 20 L/d of electrolyte-free water [10]. In this case, although the water intake was estimated as at least 15 L/d, she developed hyponatremia. The diagnosis of water intoxication was then confirmed in accordance with hypotonic hyponatremia and some specific criteria (e.g. serum osmolality < 280 mOsm/L and urine osmolality < 100 mOsm/kg) through clinical approaches [11].

The treatment of hyponatremia from water intoxication depends on the severity of the illness, and the correction of hyponatremia must be tailored to improve the patient's symptoms. Therapy typically includes fluid restriction, judicious administration of isotonic or hypertonic saline solutions and, on certain occasions, oral or intravenous urea, loop diuretics, or vasopressin receptor antagonists [1]. The administration of hypertonic saline must be judicious and care must be taken to avoid fluid overloading and the rise in serum sodium level should be monitored. A rapid increase in serum sodium concentration by 3–5 mmol/L per hour with the use of hypertonic saline is safe and efficacious in managing acute symptomatic hyponatremia [12]. Cheng et al [13] reported that early "rapid" correction of acute symptomatic hyponatremia by raising the serum sodium concentration to 15 mEq/L in 12 hours, whereas maintaining an absolute change in the serum sodium concentration of 26 mEq/L within 48 hours produced no long-term neurological sequelae in a series of male psychogenic water drinkers. A higher incidence of neurological complications would possibly be seen by a rapid correction of severe hyponatremia [14,15]. To avoid overcorrection and injuries to the brain, the correction of the plasma sodium levels at a rate slower than 25 mmol in 24 hours is suggested [11].

In conclusion, we reported a case of status epilepticus associated with hyponatremia caused by polydipsia. The metabolic imbalance of hyponatremia appeared to induce the onset of status epilepticus. Physicians in EDs should be alerted to the possibility of hyponatremia from water intoxication in patients with neurological symptoms during the observation period, particularly in those with psychiatric or psychological illness. Early detection, prompt treatment, and close monitoring of the rate of correction of the serum sodium level are crucial in preventing fatal complications.

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References

[1] Schrier RW, Bansal S. Diagnosis and management of hyponatremia in acute illness. Curr Opin Crit Care 2008;14:627–34.
[2] Verbalis JG, Goldsmith SR, Greenberg A, Schrier RW, Sterns RH. Hyponatremia treatment guidelines 2007: expert panel recommendations. Am J Med 2007;120:S1–21.
[3] Bhananker SM, Paek R, Vavilala MS. Water intoxication and symptomatic hyponatremia after outpatient surgery. Anesth Analg 2004;98:1294–6.
[4] Farrell DJ, Bower L. Fatal water intoxication. J Clin Pathol 2003;56:803–4.
[5] Cosgray RE, Hanna V, Davidhizar RE, Smith J. The water-intoxicated patient. Arch Psychiatr Nurs 1990;4:308–12.
[6] Dundas B, Harris M, Narasimhan M. Psychogenic polydipsia review: etiology, differential, and treatment. Curr Psychiatry Rep 2007;9:236–41.
[7] Yawar A, Jabbar A, Haque NU, Zuberi LM, Islam N, Akhtar J. Hyponatraemia: etiology, management and outcome. J Coll Physicians Surg Pak 2008;18:467–71.
[8] Agrawal V, Agarwal M, Joshi SR, Ghosh AK. Hyponatremia and hypernatremia: disorders of water balance. J Assoc Physicians India 2008;56:956–64.
[9] Gardner LB, Preston RA. University of Miami Division of Clinical Pharmacology Therapeutic Rounds: the water-intolerant patient and perioperative hyponatremia. Am J Ther 2000;7:23–30.
[10] Fried LF, Palevsky PM. Hyponatremia and hypernatremia. Med Clin North Am 1997;81:585–609.
[11] Moritz ML, Ayus JC. Disorders of water metabolism in children: hyponatremia and hypernatremia. Pediatr Rev 2002;23:371–80.

[12] Arieff AI, Ayus JC. Treatment of symptomatic hyponatremia: neither haste nor waste. Crit Care Med 1991;19:748–51.
[13] Cheng JC, Zikos D, Skopicki HA, Peterson DR, Fisher KA. Long-term neurologic outcome in psychogenic water drinkers with severe symptomatic hyponatremia: the effect of rapid correction. Am J Med 1990;88:561–6.
[14] Tanneau RS, Henry A, Rouhart F, Bourbigot B, Garo B, Mocquard Y, et al. High incidence of neurologic complications following rapid correction of severe hyponatremia in polydipsic patients. J Clin Psychiatry 1994;55:349–54.
[15] Hawthorne KM, Compton CJ, Vaphiades MS, Roberson GH, Kline LB. Ocular motor and imaging abnormalities of midbrain dysfunction in osmotic demyelination syndrome. J Neuroophthalmol 2009;29:296–9.